Thermoregulation during intermittent exercise in athletes with a spinal cord injury

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Abstract

Purpose: Individuals with a spinal cord injury (SCI) have impaired thermoregulatory control due to a loss of sudomotor and vasomotor effectors below the lesion level. Thus, individuals with high level lesions (tetraplegia) possess a greater thermoregulatory impairment than individuals with lower level lesions (paraplegia). Previous research has not reflected the intermittent nature and modality of wheelchair court sports, or replicated typical environmental temperatures. Hence, the purpose of this study was to investigate the thermoregulatory responses of athletes with tetraplegia and paraplegia during an intermittent sprint protocol (ISP) and recovery in cool conditions. ***Methods:*** Sixteen wheelchair athletes; 8 with tetraplegia (TP, body mass 65.2 ± 4.4 kg) and 8 with paraplegia (PA, body mass 68.1 ± 12.3 kg) completed a 60 min ISP in 20.6 ± 0.1°C, 39.6 ± 0.8% relative humidity, on a wheelchair ergometer, followed by 15 min of passive recovery. Core temperature (T_{core}), mean (T_{sk}) and individual skin temperatures were measured throughout. ***Results:*** Similar external work ($p = 0.70$, ES = 0.20), yet a greater T_{core} ($p < 0.05$, ES = 2.27) and T_{sk} ($p < 0.05$, ES = 1.50) response was demonstrated by TP during the ISP. ***Conclusions:*** Despite similar external work, a marked increase in T_{core} in TP during exercise and recovery signifies thermoregulatory differences between the groups were predominantly due to differences in heat loss. Further increases in thermal strain were not prevented by the active and passive recovery between maximal effort bouts of the ISP as T_{core} continually increased throughout the protocol in TP.

Keywords: Thermoregulatory, Intermittent Sprint Exercise, Wheelchair Sport, Tetraplegia, Paraplegia.

Introduction

Individuals with a spinal cord injury (SCI) have reduced afferent information to the thermoregulatory centre[1,2] and a loss of both sweating capacity and vasomotor control below the level of the spinal lesion.[1,3,4] As blood flow redistribution and sweating are two major thermoregulatory effectors, this suggests that individuals with a SCI have compromised thermoregulation and are at a greater risk of heat illness than able-bodied individuals.[5]

The magnitude of the thermoregulatory impairment is proportional to the level of the lesion. Exercising for 60-90 minutes at 60% $\dot{V}O_{2peak}$ in 15-25°C, trained individuals with a thoracic, lumbar or sacral SCI (paraplegia) may experience an increase in core temperature (T_{core}) similar to their able-bodied counterparts (~1°C).[5] In hot conditions (30-40°C) at the same exercise intensity, greater increases in T_{core} are demonstrated compared to the able-bodied, with even greater increases apparent in untrained individuals.[6,7] Individuals with a cervical SCI (tetraplegia) possess a smaller area of sensate skin, a lesser amount of afferent input regarding their thermal state and a reduced efferent response compared to individuals with paraplegia.[4,8] Less is known regarding the thermoregulatory responses of athletes with tetraplegia during exercise. Yet, it is thought they may experience a disproportionate increase in T_{core} and heat storage, due to the presence of little or no sweating response, leading to a greater degree of thermal strain.[9] Price and Campbell[9] demonstrated that an athlete with tetraplegia arm cranking at 60% $\dot{V}O_{2peak}$ for 60 minutes in ~21.5°C, experienced a continuous increase in T_{core}, in contrast to a plateau experienced by able-bodied and athletes with paraplegia. While the athlete with tetraplegia did not experience high thermal strain in these conditions, the continuous rise in T_{core} shows that thermal balance was not achieved.

Previous research has predominantly used arm cranking protocols[6,10] to examine the thermoregulation of athletes with a SCI and not their habitual mode of wheelchair exercise. However, thermoregulatory differences exist between different modalities with lower physiological and thermal strain elicited during wheelchair propulsion due to intermittent application of force to the flywheel, compared to continuous force application during arm cranking.[11] Moreover, previous studies have not matched the ambient conditions to indoor playing environments or the intermittent nature of wheelchair court sports, such as wheelchair basketball and rugby. Therefore, the purpose of this study was to compare the thermoregulatory responses of athletes with paraplegia and tetraplegia during intermittent sprint wheelchair exercise and recovery in cool conditions.

Methods

Participants

Eight wheelchair rugby players with tetraplegia (TP: 7 males, 1 female, 1 incomplete lesion)[12] and eight wheelchair basketball players with paraplegia (PA: 7 males, 1 female, 3 incomplete lesions)[12] (Table 1), gave their written informed consent to participate in this experimental research study. The study was approved by the University Research Ethics Committee and was conducted in accordance with the Declaration of Helsinki.

Insert Table 1 here

Preliminary tests

On arrival at the laboratory, skinfold measurements were taken from the following sites; biceps, triceps, subscapular, superilliac and abdomen followed by a continuous incremental test on a treadmill to determine peak oxygen uptake ($\dot{V}O_{2peak}$). For the $\dot{V}O_{2peak}$ test, workload increased by 0.2 or 0.3 m/s every 3 min (dependent on the individual's classification) until the participant could no longer maintain the speed of the treadmill.

Experimental Conditions

Participants ingested a telemetry pill (HQ Inc, Palmetto, Florida), ~8 h prior to the start of the test, for the measurement of core temperature (T_{core}). Two hours after the preliminary test, participants were weighed (Marsden Weighing Group Limited, Henley-on-Thames, UK) with no clothing covering their upper body. During the intermittent sprint protocol (ISP) participants wore their usual training attire of lightweight tracksuit trousers and either a short or long sleeved top. Seven skin thermistors (Grant Instruments, Cambridge, UK) were placed on the right side of the body on the forehead, forearm, biceps, upper back, chest, thigh and calf for measurement of skin temperature (Grant Squirrel logger, Series 2010,Grant Instruments, Cambridge, UK). Mean skin temperature (T_{sk}) was estimated in accordance with the formula by Ramanathan.[13] Heat storage (HS) was calculated using the following formula:[14]

$$\text{Heat storage} = (0.8\ \Delta T_{core} + 0.2\Delta T_{sk})\cdot c_b$$

where c_b is the specific heat capacity of the body tissue (3.49 $J\cdot g^{-1}\cdot °C^{-1}$) and ΔT_{core} and ΔT_{sk} represent changes in T_{core} and T_{sk} from rest to the end of each exercise block and recovery. An estimate of external work was calculated by total distance covered (m) during the ISP multiplied by total resistance (N) of the wheelchair ergometer-wheelchair system.

Following instrumentation and transfer to their own sports wheelchair participants rested for 10 min, before completing a self-selected warm-up on a single cylinder wheelchair ergometer (WERG, Bromakin, Loughborough, UK).[15] During the warm-up, participants performed a deceleration test for power and resistance to be calculated.[16]

The ISP was conducted in an environmental chamber at 20.6 ± 0.1°C and 39.6 ± 0.8% relative humidity chosen to replicate a sports hall environment. All participants completed the test at a similar time in the afternoon to negate circadian variation and refrained from caffeine and alcohol 24 h before the test. The ISP simulated an on-court session and is reported elsewhere.[17] Briefly, the ISP consisted of four exercise blocks separated by 4.5 min of passive recovery (Figure 1). Each block comprised of six bouts of 30 s, where athletes performed alternate three pushes forwards and backwards for the first 15 s followed by a 15 s sprint at maximum effort. Bouts were followed by 90 s of active recovery of low intensity. At the end of block four, participants rested for 15 min before all thermistors were removed and the participant was re-weighed. The whole session lasted 55.5 min with maximum intensity activity accounting for 12 min, including a total of 24 sprints. Verbal encouragement was given throughout the test.

Insert Figure 1 here

Heart rate (HR) was recorded at 5 s intervals during the ISP (Polar PE 4000, Kempele Finland). Whole body ratings of perceived exertion (RPE)[18] and thermal sensation[19] were recorded at the end of each exercise block. Prior to the start of the ISP and during recovery thermal sensation was also recorded. The thermal sensation scale, comprised of categories ranging from 0 (“unbearably cold”) to 8 (“unbearably hot”). After the warm-up and upon completion of exercise capillary blood samples from the earlobe were taken and analysed for haematocrit (Haemtospin 1300, Hawksley, Lancing, UK) and haemoglobin (B-Hemoglobin, Hemocue Limited, Dronfield, UK) to determine plasma volume.[20] Capillary blood samples were taken at the end of each block for analysis of blood lactate (BLa) concentration (YSI SPORT, YSI Incorporated, Ohio, USA). Participants were allowed to drink *ad libitum* during the passive recovery between blocks.

Statistical Analysis

All data was checked for normality, using the Shapiro–Wilk test. Delta core and skin temperatures were calculated. Independent t-tests were used to analyse any between group differences in participant characteristics, total distance, total resistance, external work, fluid balance and start and end T_{core}, T_{sk} and HS. Sprint speed and power output across the 24 sprints, physiological and thermoregulatory responses were analysed using a two way (group x time) analysis of variance (ANOVA). Where significance was obtained post-hoc pairwise comparisons with a Bonferroni correction were conducted. For individual skin temperatures and heat storage during recovery data from seven TP were used, as data from the last three minutes of recovery were missing for one participant. For all comparisons where the assumption of sphericity was violated, a Greenhouse–Geisser correction was applied. Effect sizes (ES) were estimated by Cohen's d, where 0.2 represented a small effect size, 0.5 a medium effect size, and 0.8 a large effect size.[21] All data were analysed using SPSS version 19.0 and significance was accepted at the $p \leq 0.05$ level.

Results

Participant characteristics

There were no differences between TP and PA for the physiological and participant characteristics ($p>0.05$, Table 1). Yet, large effect sizes were apparent for $\dot{V}O_{2peak}$ (ES = 0.89) and training hours per week (ES = 0.73).

Sprint performance

There were no differences between groups or across the 24 sprints for either sprint speed or peak power output (all $p>0.05$, Table 2). Total resistance of the wheelchair ergometer-wheelchair system was greater in TP ($p = 0.01$, ES = 1.64) whilst total distance covered during the ISP was greater for PA

($p < 0.001$, ES = 1.92). External work was not statistically different between groups ($p = 0.70$, ES = 0.20).

Insert Table 2 here

Physiological responses

Mean and peak HR for each block of the ISP were greater for PA than TP ($p < 0.05$, Table 3). Mean HR for both groups increased from block 1 to 2 then remained stable throughout exercise. For both groups peak HR was similar over time ($p = 0.43$). Throughout exercise BLa was similar over time ($p = 0.09$) but different between groups (8.08 ± 3.04 and 8.73 ± 2.17 for TP and PA, respectively, $p = 0.02$, ES = 0.25).

Insert Table 3 here

Core temperature

Core temperature was similar between groups at the start of exercise (37.0 ± 0.6°C and 37.1 ± 0.3°C for TP and PA, respectively, $p = 0.75$, ES = 0.16). At the end of exercise TP demonstrated a greater T_{core} than PA (38.2 ± 0.5°C and 37.6 ± 0.4°C for TP and PA, respectively, $p = 0.02$, ES = 1.32). During both exercise and recovery, TP experienced a greater increase in T_{core} from resting values than PA (both $p < 0.0001$, ES = 0.75 and ES = 2.27 for exercise and recovery, respectively, Figure 2). At the end of recovery, T_{core} for TP remained elevated from rest by 1.1°C compared to 0.2°C for PA (38.1 ± 0.5°C and 37.3 ± 0.3°C for TP and PA, respectively, $p < 0.001$, ES = 1.84).

Skin temperature

Mean skin temperature was similar between groups at the start (29.5 ± 1.6°C and 30.6 ± 0.6°C for TP and PA, respectively, $p = 0.09$, ES = 0.91) and end of exercise (30.2 ± 1.5°C and 30.0 ± 1.6°C for TP and PA, respectively, $p = 0.75$, ES = 0.16) and end of recovery (30.0 ± 1.4°C and 29.7 ± 1.8°C for TP and PA, respectively, $p = 0.76$, ES = 0.16). During exercise and recovery the change in T_{sk} from resting values was different between TP and PA ($p < 0.001$, ES = 1.50, $p = 0.02$, ES = 1.43 for exercise and recovery, respectively). For the PA group, T_{sk} decreased during exercise whilst athletes with TP experienced an increase in T_{sk} (Figure 2). Individual skin temperatures (Figure 3) were similar between groups at the start and end of exercise ($p>0.05$). During exercise, back skin temperature was the only site that demonstrated a difference between groups with an increase from resting values in TP (0.9 ± 0.6°C) and a decrease in PA (-0.4 ± 0.9°C, $p < 0.001$, ES = 1.65). During recovery, chest, back, forearm and forehead skin temperature remained elevated from start of recovery values to a greater extent in TP than PA ($p<0.05$).

Insert Figure 2 here

Insert Figure 3 here

Heat storage

Heat storage was greater in TP (2.8 ± 1.2 $J \cdot g^{-1}$) than PA (1.0 ± 1.0 $J \cdot g^{-1}$) during exercise (Figure 4, $p < 0.001$, ES = 1.61) and at the end of recovery (3.4 ± 1.4 $J \cdot g^{-1}$ and -0.5 ± 1.3 $J \cdot g^{-1}$ for TP and PA, respectively, $p < 0.001$, ES = 3.08).

Insert Figure 4 here

Perceptual measures

During exercise RPE was similar between groups ($p = 0.52$, ES = 0.24) with an increase over time (14 ± 1 and 16 ± 2 for the end of block 1 and 4, respectively). Thermal sensation was similar between groups during exercise, (4 ± 1 and 6 ± 1 at rest and end of block 4, respectively, $p = 0.29$, ES = 0.31) and recovery (6 ± 1 and 3 ± 1 at the start and end of recovery, respectively, $p = 0.69$, ES = 0.14).

Fluid balance

Both TP and PA drank similar amounts during the ISP and recovery (540 ± 112 ml and 469 ± 233 ml for TP and PA, respectively, $p = 0.45$, ES = 0.39). The change in body mass (0.4 ± 0.4 kg and 0.1 ± 0.3 kg for TP and PA, respectively, $p = 0.11$, ES = 0.84) and plasma volume changes were similar between groups (4.0 ± 13.7% and 4.3 ± 9.5% for TP and PA, respectively, $p = 0.96$, ES = 0.03).

Discussion

The main findings indicate that despite external work being similar between groups, T_{core} and HS increased at a greater magnitude in TP compared to PA during intermittent sprint exercise in cool conditions. The greater increase in T_{core} for TP signifies that thermoregulatory differences between the groups were predominantly due to a lower capacity for heat loss in TP compared to PA. Even during post-exercise recovery T_{core} and HS remained elevated in TP signifying an inability to dissipate the heat produced during exercise resulting in the retention of heat during recovery.

Further increases in thermal strain in TP were not prevented by the active and passive recovery between the maximum effort bouts as T_{core} and HS were found to continually increase throughout the protocol in this group. The T_{core} responses for both groups are therefore comparable to previous studies during continuous wheelchair exercise, with increases of 0.2-0.7°C[6,22] and 0.9°C[9] observed for PA and TP, respectively.

The mean skin temperature response of the two groups likely reflects the athletes' sweating capacity, being proportional to lesion level. For instance, the greater reduction in sweating capacity in TP resulted in an increase in T_{sk} during exercise. In PA, T_{sk} decreased during exercise, likely due to the larger body surface area available for sweating and therefore greater evaporative cooling of the skin. It should be noted that although T_{sk} was not significantly different at the onset of exercise, a large ES demonstrates PA may have had a substantially warmer starting T_{sk} than TP. Yet, mean skin temperature data should be interpreted with caution in individuals with a SCI as it may mask regional skin temperature responses.[5]

During exercise, differing responses in back skin temperature were apparent, increasing in TP and decreasing in PA, due to the majority of the upper body skin of TP being insensate compared to sensate in PA. Yet a similar finding was not found for chest skin temperature. Sweat rates vary with body region in able-bodied individuals, with a greater sweat rate apparent at the upper back than the chest.[23] Therefore, at the chest, a lower evaporative cooling effect of sweat may have been apparent in PA, resulting in a chest skin temperature similar to that seen in TP. In both groups, upper arm skin temperature demonstrated a decrease during exercise shown previously, yet more pronounced, during continuous wheelchair propulsion.[11] The decrease in upper arm skin temperature is thought to be caused by the arm moving relative to the body in wheelchair propulsion causing convective cooling to the upper arm.[11]

Neither group experienced a change in thigh skin temperature during exercise or recovery likely due to the disrupted blood flow and vascular atrophy below the level of the lesion.[3] Although small, there was a significant increase from rest in calf skin temperature over time, possibly due to the variable response of calf skin temperature in PA.[10] A greater increase in calf skin temperature, than the present study, has been previously observed during prolonged arm cranking leading the authors to suggest the lower body is a potential site for HS in PA.[9,10] The degree of sweating and blood flow redistribution in the lower limb may be dependent on the lowest intact part of the sympathetic chain, with the pathway for vasodilation in the lower limb located at or below T10.[22] In individuals with lesions at T12, calf skin temperature has been shown to increase during exercise with little or no change for individuals with lesions at T10/T11.[22] However, in the present study, similar trends in calf skin temperature were apparent for individuals with lesions above (n =5) and below T10 (n=3) in the PA group. To fully understand the underlying mechanisms of vasomotor control of the lower body during upper body exercise further study is required.

More pronounced differences between skin temperature sites may have been masked by the large inter-individual variations in skin temperatures, a noticeable response in studies in the SCI population.[24,25] These variations may have been heightened by the large range of lesion levels in PA (T4-S1), resulting in differences in sympathetic and somatosensory pathways, in arrangements of sympathetic outflow and the type and degree of reinnervation.[3,10]

From a perceptual perspective, even though TP were exercising at a greater T_{core} than PA, similar thermal sensation scores throughout exercise indicate they did not perceive to be warmer. This may be related to training status with potentially a greater T_{core} being better tolerated by the highly trained. Although not significant a large ES in training hours (ES = 0.73) signifies the group of TP in the present study were more highly trained, hence may have a better tolerance of greater T_{core} values. Due to the smaller surface area of sensate skin in TP compared to PA, it is also possible that TP may not perceive the increase in body temperature as effectively.[26] During higher intensity exercise and in warmer ambient conditions this may be of more concern, especially as these athletes could potentially override perceived signs of thermal strain, putting them at risk of heat illness.[26]

The training status of TP may have led to a greater development of their remaining musculature.[27] Potentially, this may have enabled TP to produce similar power outputs and external work to PA. The larger total resistance of the wheelchair ergometer-wheelchair system for TP was, however, likely caused by the differences in the mass of the wheelchairs used in wheelchair basketball and rugby, with heavier wheelchairs used in the latter (~11-13 kg vs. 15-19 kg). The lower mean and peak HR in TP, due to the reduced sympathetic innervation of the heart, is consistent with previous studies.[28] Although there was no significant difference in $\dot{V}O_{2peak}$, a large ES signifies a meaningful difference between the groups was apparent, with previous research indicating an inverse relationship exists between lesion level and $\dot{V}O_{2peak}$.[28] The extent to which the athlete's aerobic fitness would have affected the results is unclear, yet, future work matching the groups for training status may accentuate the differences in thermoregulatory responses due to the level of spinal lesion.

Practical Applications

Although neither group were under considerable thermal strain, the present study highlights that TP experience a greater increase in T_{core} for the same external work load of intermittent sprint exercise compared to PA. Even though the protocol had greater ecological validity than previous studies due to the intermittent nature and use of wheelchair propulsion, the ISP may not have been wholly reflective of a wheelchair basketball or rugby match. Total distances covered were considerably shorter (2316 m) than the activity profiles of wheelchair rugby players during a match (4540 m).[29] If the ISP was of a similar magnitude to match play, i.e. greater metabolic work, the athletes may have experienced a greater thermal response, especially TP. Practically, support staff should closely monitor TP for signs of heat stress during wheelchair court sports, and if possible, apply appropriate cooling before, during or following play.

A limitation of the study may be the inclusion of four individuals with an incomplete SCI (one TP and three PA) in the mean group values. The degree of autonomic dysfunction may be dependent on the completeness of the injury,[28] with incomplete lesions resulting in a greater amount of sensory information regarding their thermal state and a greater capacity to sweat.[26] Nevertheless, their inclusion was justified as their T_{core} and T_{sk} responses were within one standard deviation of the mean response of each group.

Conclusion

Similarly to continuous arm cranking and wheelchair exercise, TP have a greater inability to dissipate heat than PA during intermittent sprint exercise in cool conditions. Despite the two groups producing similar amounts of external work, TP had a marked increase in T_{core} during exercise and recovery, signifying that differences between the groups were predominantly due to differences in heat loss. Neither group were under high levels of thermal strain yet the present study highlights the heightened thermal response of TP to intermittent wheelchair exercise, with caution that a greater T_{core} response may be apparent during actual game play. Support staff should be aware of the greater thermal impairment experienced by TP in wheelchair court sports, monitoring them for signs of heat stress, and if possible, apply appropriate cooling before, during or following play.

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Figure legends

Figure 1 - Schematic of the intermittent sprint protocol (ISP), including all measures taken throughout the four exercise blocks and recovery. The black blocks depict both the 15 s of alternate forwards and backwards pushing and the 15 s sprints. The white blocks depict the 90 s of active recovery. The grey blocks show the 4.5 min of passive recovery between each exercise block and the 15 min of recovery following the ISP. The corresponding exercise blocks and recovery periods are numbered below the time axis and Figures 2-3 will refer to these labels (E = exercise block, R = passive recovery). Warm–up is not included in the Figure. TS = thermal sensation, PV = measures to determine plasma volume (haemoglobin and haematocrit), BLa = blood lactate, RPE = rating of perceived exertion.

Figure 2 - Change in core temperature ($T_{core,}$ A) and mean skin temperature (T_{sk} , B) from resting values during exercise and recovery for athletes with tetraplegia (TP) and athletes with paraplegia (PA) during each exercise block and recovery (E = exercise block, R= passive recovery). *significantly different from PA ($p<0.05$).

Figure 3 - Individual skin temperatures (A-back, B-upper arm, C-calf, D-thigh) for athletes with tetraplegia (TP) and athletes with paraplegia (PA) during each exercise block and recovery (E = exercise block, R = passive recovery). *significantly different from PA ($p<0.05$).

Figure 4 - Heat storage for athletes with tetraplegia (TP) and athletes with paraplegia (PA) during each exercise block and recovery. *significantly different from PA ($p<0.05$).

Table 1 Physiological and participant characteristics of athletes with tetraplegia (TP) and paraplegia (PA) (Mean ± S.D.)

	TP	PA
Age (years)	27.4 ± 4.2	27.8 ± 6.2
Body mass (kg)	65.2 ± 4.4	67.7 ± 13.1
Sum of skinfolds (mm)	65.4 ± 28.2	78.2 ± 38.2
$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	1.55 ± 0.37	1.92 ± 0.47
Lesion level (range)	C4/5-C6/7	T4-S1
Time since injury (years)	8.0 ± 4.6	11.4 ± 7.7
Training ($h \cdot week^{-1}$)	15.0 ± 4.2	11.0 ± 6.4

Table 2 Sprint performance for athletes with tetraplegia (TP) and paraplegia (PA) (Mean ± S.D.)

	TP	**PA**
Sprint speed (m/s)[a]	3.14 ± 0.59	3.51 ± 0.44
Peak power output (W)[a]	67 ± 14	59 ± 14
Total resistance (N)	21 ± 3*	17 ± 3
Total distance (m)	2316 ± 258*	3042 ± 468
External Work (kJ)	49 ± 5	51 ± 9

[a] Sprint speed and power output across the 24 sprints. *significantly different from PA ($p<0.05$).

Table 3 Mean and peak heart rate (HR) and blood lactate during the intermittent sprint protocol (ISP) for athletes with tetraplegia (TP) and paraplegia (PA) (Mean ± S.D.)

	TP	PA
Mean HR (beats min^{-1})	107 ± 6*	132 ± 15
Peak HR (beats min^{-1})	133 ± 6*	161 ± 8
Blood lactate (mmol/l)	8.08 ± 3.04*	8.73 ± 2.16

*significantly different from PA ($p<0.05$).

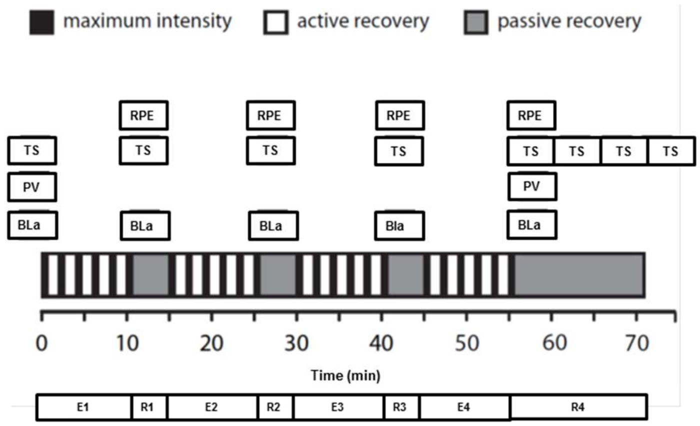

Figure 1 - Schematic of the intermittent sprint protocol (ISP), including all measures taken throughout the four exercise blocks and recovery. The black blocks depict both the 15 s of alternate forwards and backwards pushing and the 15 s sprints. The white blocks depict the 90 s of active recovery. The grey blocks show the 4.5 min of passive recovery between each exercise block and the 15 min of recovery following the ISP. The corresponding exercise blocks and recovery periods are numbered below the time axis and Fig. 2-3 will refer to these labels (E = exercise block, R = passive recovery). Warm–up is not included in the Fig. TS = thermal sensation, PV = measures to determine plasma volume (haemoglobin and haematocrit), BLa = blood lactate, RPE = rating of perceived exertion
254x190mm (96 x 96 DPI)

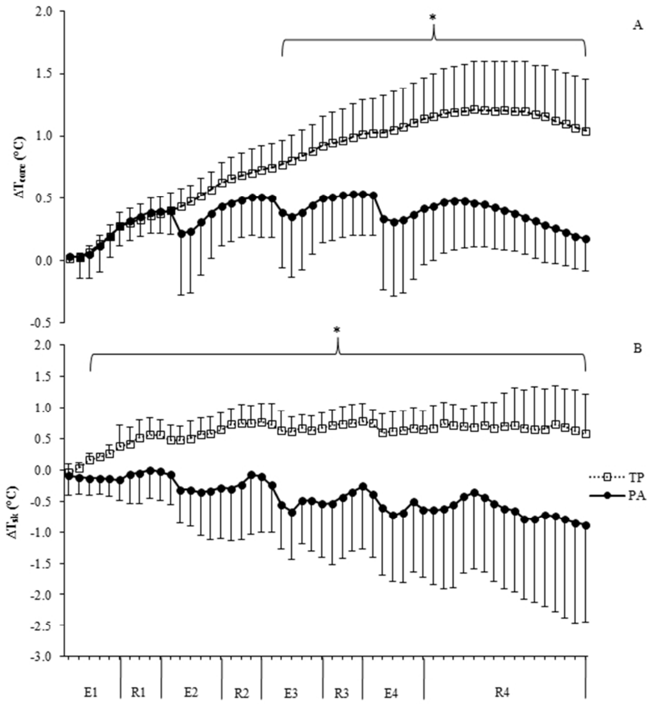

Figure 2 - Change in core temperature (Tcore, A) and mean skin temperature (Tsk , B) from resting values during exercise and recovery for athletes with tetraplegia (TP) and athletes with paraplegia (PA) during each exercise block and recovery (E = exercise block, R= passive recovery). *significantly different from PA ($p<0.05$)
254x190mm (96 x 96 DPI)

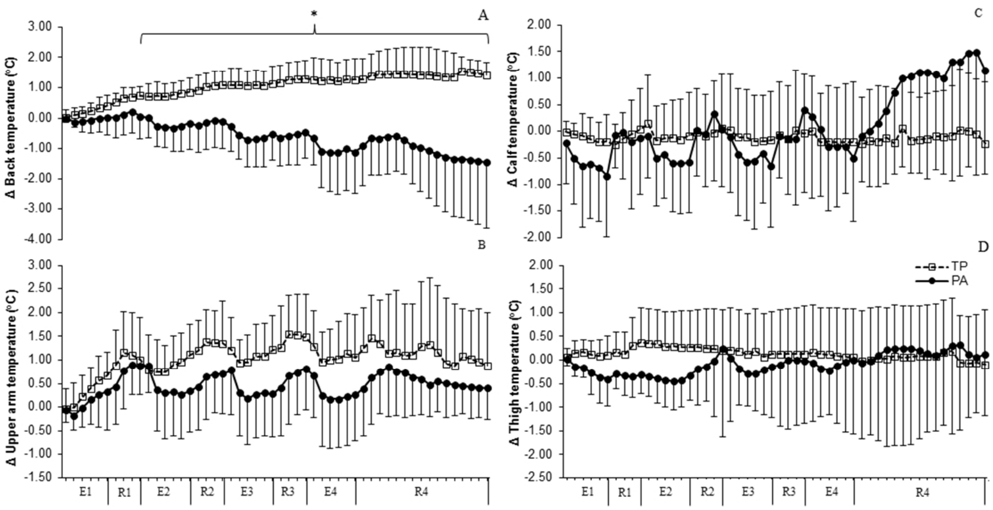

Figure 3 - Individual skin temperatures (A-back, B-upper arm, C-calf, D-thigh) for athletes with tetraplegia (TP) and athletes with paraplegia (PA) during each exercise block and recovery (E = exercise block, R = passive recovery). *significantly different from PA ($p<0.05$)
275x190mm (96 x 96 DPI)

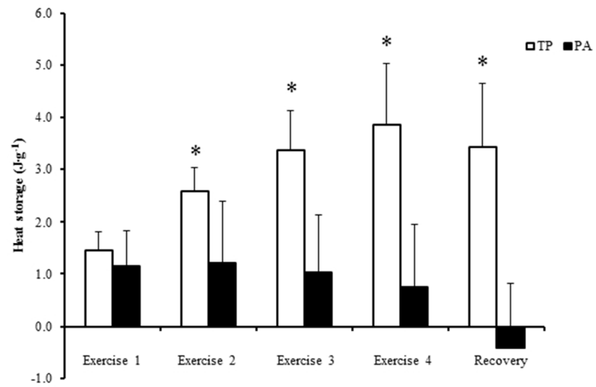

Figure 4 Heat storage for athletes with tetraplegia (TP) and athletes with paraplegia (PA) during each exercise block and recovery. *significantly different from PA ($p<0.05$)
254x190mm (96 x 96 DPI)